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CERTIFICATE OF MAILING UNDER 37 C.F.R. §1.8(a)

The undersigned hereby certifies that this document is being placed in the United States mail with first-class postage attached, addressed to MAIL STOP AMENDMENT, Commissioner for Patents, P.O. Box 1450, Alexandria, VA 22313-1450, on April 21, 2006.

MAIL STOP AMENDMENT
Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

Sir:

DECLARATION OF BARRY JONES UNDER 37 CFR §1.132

I, Barry Jones, declare as follows:

1. I am a co-inventor on the above-identified application. I make this Declaration in support of the Amendment filed in connection with the above-identified application, and in response to the Office Action dated October 21, 2005.

2. I am the Chief Scientific Officer at Point Therapeutics, Inc. Point Therapeutics, Inc. is the sole assignee of the above-identified application. My curriculum vitae is attached to this Declaration.

3. This Declaration describes experiments disclosed in the above-identified application and the results of these experiments. The experiments and results are described at least in Example 4 and Fig. 3 of the specification.

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4. Immunodeficient NOD/SCID mice were inoculated with Namalwa cells derived from a Burkitt's Non-Hodgkin's lymphoma sample on day 1. The human lymphoma cells proliferated in the immunodeficient mice to form solid subcutaneous tumors. Mice were administered 1.5 mg of normal human IgG or 1.5 mg of the human anti-CD20-specific antibody rituximab on each of days 3, 5 and 7 after tumor inoculation (indicated as triangles on Fig. 3). Mice were also administered 5 μ g of the Formula I agent PT-100 (i.e., Val-boroPro) twice daily from day 2 until day 20 after tumor inoculation. The four treatment groups each contained 4 or 5 replicate mice. The data represent mean tumor volumes (+/- SE).

5. Fig. 3 is a graph showing the effect of control IgG, PT-100 and control IgG, anti-CD20 antibody rituximab alone, and PT-100 and anti-CD20 antibody rituximab together on tumor volume as function of time after inoculation. Control treatment with normal human IgG had no effect on tumor growth as compared with saline treatment (data not shown). Treatment with PT-100 and normal human IgG or with rituximab alone each significantly ($p<0.05$) inhibited tumor growth. Combined treatment with PT-100 and rituximab inhibited tumor growth to a significantly ($p<0.05$) greater extent than did treatment with PT-100 and normal human IgG or treatment with rituximab alone.

6. These experiments and data correlate with the claimed methods in the above-identified application which relate to enhancing anti-CD20 antibody treatment in a subject having cancer by administering the antibody in conjunction with an agent of Formula I. PT-100 is an agent of Formula I, and rituximab is an anti-CD20 antibody.

7. As stated in the above-identified application, the results show that an agent of Formula I (e.g., PT-100) enhances the anti-tumor effect of an anti-CD20 antibody (e.g., rituximab). The combination of the agent of Formula I with the antibody therefore has unexpected benefit. It was unpredictable based on the knowledge in the art prior to the invention that PT-100 could enhance the anti-tumor effect of an antibody such as an anti-CD20 antibody at least due to the difference in their mechanisms of action as understood at the time. The anti-cancer effect of antibodies such as anti-CD20 antibodies is mediated via an immunological mechanism of action. It was not

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appreciated, prior to the invention, that Formula I agents such as PT-100 could also induce an anti-cancer effect via an immunological mechanism. Therefore the ability of these latter agents to enhance the efficacy of an immunologically acting anti-cancer antibody could not have been predicted and thus was unexpected.

8. I, the undersigned, declare that all statements made herein of my own knowledge are true and that all statements made on information and belief are believed to be true. And further, that the statements were made with the knowledge that willful false statements and the like so made are punishable by fine or imprisonment, or both, under §1001 of title 18 of the United States code and that such willful false statements may jeopardize the validity of this document and any patent which may issue from the above-identified patent application.

APRIL 21, 2006

Date



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SUMMARY

- Immunologist with 15 years of experience in biotechnology management and research, including 6 years in senior management, backed by more than ten years of academic research.
- Proven expertise in executing research projects and managing multidisciplinary programs. Strong record of implementing research strategies that facilitate goal achievement as demonstrated by successful negotiation and management of corporate research collaborations and publication in peer-reviewed journals.
- Recognized for leadership abilities, recruiting and retaining qualified staff, strong verbal and written skills, and depth of knowledge in immunology relevant to human disease and drug discovery.

PROFESSIONAL EXPERIENCE

Point Therapeutics, Boston, MA, 08/97-present

Chief Scientific Officer 04/2006-present

Senior Vice President, Research 01/2003-present

Member of Senior Management Team and an Officer of the Company. Responsible for integrating research programs with business goals. Experience in translation of basic research into clinical applications. Overall responsibility for research in-house. Provided preclinical research that successfully supported clinical development in oncology of lead product candidate, talabostat (PT-100), through Phase 1 and into Phase 2.

Vice President, Research, 01/2000-12/2002

Management of research in tumor immunology and hematopoiesis in animal models and human *in vitro* systems that provided 'proof of concept' for small molecules acting to stimulate hematopoiesis and immunity. The research promoted corporate partnership, financing, a Phase I Safety Trial in U.K. and IND filing in U.S.

Director Research, 08/97-01/2000

Management, design and hands-on implementation of research projects.

Procept, Inc., Cambridge, MA, 1991-1997.

Director of Immunology, 1993-07/97

Managed department performing cellular and molecular research for programs in T-cell receptors, co-receptors and immunosuppressive drugs. Core expertise included monoclonal antibody production, flow cytometry, and animal research for investigation of models of autoimmune disease and allograft rejection. Major additional responsibilities were:

Program leader of the T-cell receptor program reporting to corporate partner, Bristol-Myers Squibb. T-cell receptor structure and application of recombinant soluble T-cell receptors in immunotherapy of autoimmune disease were investigated by an inter-departmental team of 20 scientists. Research has been published and presented at international scientific meetings.

Co-program leader for the small-molecule immunosuppressive drug program evaluating compounds in assays of T-cell function and animal models of graft rejection and autoimmune disease.

Scientific steering committee member for the CD4/CD2 program with Sandoz (now Novartis), Basle, Switzerland).

Principal Investigator II, Immunology, 1991-1993

Initiated and lead the autoimmune disease project in the T-cell receptor program. Conducted experiments investigating autoimmune T-cells in: NOD mouse model of type 1 diabetes, and experimental allergic encephalomyelitis of rat and mouse.

Pennsylvania State University, State College, PA, 1988-1991

Assistant Professor, Department of Molecular and Cell Biology, 1988-1991.

Directed an immunology laboratory with responsibility for research and training of graduate students in cellular and molecular immunology. Taught classes in immunology and genetic basis of human disease.

Yale University, New Haven, CT, 1978-1988

Senior Associate, Howard Hughes Medical Institute, 1983-1988

Research Associate, Department of Pathology (Immunology Division), 1978-1982

EDUCATION

Post-Doctoral Research in Tumor Immunology, 1974-1978, Middlesex Hospital Medical School, London, UK.

Ph.D. in Immunology, 1974, Bristol University, Bristol, UK

B.Sc. (First Class Honors), 1971, Liverpool University, Liverpool, UK

PUBLICATIONS

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9. Adams, S. and Jones, B. Characterization of innate effector cells involved in the anti-tumor activity of talabostat (PT-100). AACR 97th Annual Meeting Proceedings, 2006; Vol. 47 (abstr. 4659).

Patents

Hematopoietic stimulation. 2001 Inventors: Wallner, B. P., Jones, B., Miller, G. T., and Adams, S. U.S. Patent 6,300,314-Issued 10/09/01